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The Role of Dopamine Deficiency, REM Sleep Behavior Disorder, and Hyposmia in Cognitive Decline in Early Neuronal Synucleinopathy

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Description

Dopamine deficiency and REM sleep behavior disorder (RBD) are hallmark features of several neurodegenerative conditions, including early neuronal synucleinopathies, such as Parkinson's disease and dementia with Lewy bodies. These conditions are associated with the abnormal accumulation of alpha-synuclein protein in the brain, which disrupts normal neuronal functioning. One of the earliest symptoms often noted in these diseases is hyposmia, or reduced sense of smell, which frequently precedes more overt cognitive and motor impairments. As the disease progresses, cognitive decline becomes more prominent, and understanding the interplay between dopamine deficiency, RBD, and hyposmia is essential to understanding the broader impact on cognitive function in these early stages. Dopamine is a neurotransmitter crucial for regulating mood, movement, and cognition. In synucleinopathies, the degeneration of dopaminergic neurons, particularly in the substantia nigra, leads to a profound deficiency of dopamine in several brain regions, including the striatum. Dopamine deficiency directly affects cognitive processes such as attention, memory, and executive function. In early stages of the disease, individuals may experience subtle cognitive impairments, such as difficulty with concentration and decision-making, without the more classic signs of dementia. This impairment is partly due to the reduced ability of the brain to coordinate the activities necessary for complex cognitive tasks, which rely heavily on dopamine signaling. As the disease progresses, the cognitive deficits often become more pronounced, leading to difficulties in problem-solving, planning, and managing day-to-day activities. In addition to dopamine deficiency, REM sleep behavior disorder (RBD) is a common feature in individuals with early synucleinopathies. RBD is characterized by the loss of muscle atonia during REM sleep, causing individuals to physically act out their dreams, which can result in violent movements and sleep disturbances. This condition is thought to reflect the pathological changes occurring in the brainstem and can be an early indicator of synucleinopathy, sometimes even preceding motor symptoms. The presence of RBD has been linked to increased cognitive decline in individuals with synucleinopathies. Disrupted sleep patterns and the frequent awakening associated with RBD contribute to sleep deprivation, which in turn can exacerbate cognitive deficits. Poor sleep quality and insufficient restorative sleep negatively affect memory consolidation and cognitive function, further accelerating the progression of the disease. Hyposmia, or a reduced sense of smell, is another early symptom of synucleinopathies, often occurring well before motor or cognitive symptoms become clinically significant. The olfactory system, which involves areas of the brain such as the olfactory bulb and the entorhinal cortex, is one of the first regions affected by the accumulation of alpha-synuclein. Hyposmia can be an early warning sign of neuronal synucleinopathy, with studies showing that individuals with reduced smell perception are at higher risk for developing Parkinson's disease or related conditions. The loss of smell may also correlate with changes in cognitive function, as the olfactory system shares neural pathways with areas of the brain involved in memory and learning. While hyposmia itself may not directly cause cognitive decline, it is considered an important early biomarker of neurodegeneration and may help identify individuals at higher risk of developing more severe cognitive impairments.The combination of dopamine deficiency, RBD, and hyposmia has a significant cumulative effect on cognition in the early stages of neuronal synucleinopathies. Dopamine deficiency contributes to cognitive dysfunction by impairing attention, executive function, and memory, while RBD exacerbates these deficits by disrupting sleep and reducing the brain's ability to consolidate memories. Additionally, hyposmia serves as an early indicator of the disease, alerting clinicians to the possibility of underlying neurodegeneration before cognitive and motor symptoms fully manifest. Together, these factors contribute to a more complex cognitive profile in individuals with synucleinopathies, which can complicate early diagnosis and intervention. The impact of these early signs on cognition emphasizes the need for early diagnostic tools that can detect changes in dopamine levels, sleep patterns, and olfactory function. Identifying these early symptoms and understanding their interconnectedness may provide crucial insights into the progression of the disease and allow for earlier, more targeted interventions. While there is no cure for synucleinopathies, early identification and management of these symptoms can help slow the progression of cognitive decline and improve the quality of life for affected individuals.

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Conflict of Interest

Authors declare that they have no conflict of interest.

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